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Physical Symptom Trajectories Following Trauma Exposure: Longitudinal Findings from the Normative Aging Study

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This study modeled physical symptom trajectories from ages 30 to 75 in 1079 older male military veterans who were assessed every 3 to 5 years since the 1960s. Combat exposure and noncombat trauma were used to define four groups: no trauma (N = 249), noncombat trauma only (N = 333), combat only (N = 152), and both combat and noncombat trauma (N = 345). Number of symptoms on the Cornell Medical Index physical symptom scale increased 29% per decade. Men who had experienced either combat or noncombat trauma did not differ from nonexposed men, but those who had experienced both combat and noncombat trauma had 16% more symptoms across all ages. There were no differences in age-related trajectories as a function of trauma history. In cross-sectional analysis, men with combat and noncombat trauma had more posttraumatic stress disorder symptoms, but not more depression symptoms, than men with either no trauma or noncombat trauma only. Discussion focuses on the importance of considering physical as well as psychological outcomes of exposure to traumatic events.

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An increasing amount of evidence indicates that exposure to a traumatic event is linked to poor physical health outcomes. Golding and colleagues (1997) recently reported a meta-analysis of seven studies of the relationship between sexual assault history and health perceptions. Sexual assault was associated with a 46% increased likelihood of poor subjective health, even when the effects of depression were controlled. The association between trauma and physical health is not limited to either sexual trauma or self-reports only, however. Friedman and Schnurr (1995) reported in a qualitative literature review that military trauma, sexual trauma, and natural or human-made disasters, as well as other types of trauma, were linked to poor outcomes in four domains of physical health: self-reported health and symptoms (*e.g.*, Kulka et al., 1990; Wolfe et al., 1994), utilization (*e.g.*, Bergman and Brismar, 1991; Kimerling and Calhoun, 1994), morbidity as indicated by physician diagnosis (*e.g.*, Leserman et al., 1996; Sibai et al., 1989), and, to some extent, mortality (*e.g.*, Adams and Adams, 1984).

Adverse physical health outcomes can be observed long after a traumatic event has occurred. For example, in adult women, there is an association between childhood sexual abuse and gastrointestinal disorder (Leserman et al., 1996), chronic pelvic pain (Walker et al., 1992), and other physical conditions (Rimsza and Berg, 1988). Very long-term adverse outcomes have been demonstrated in studies of prisoners of war (Goulston et al., 1985) and of concentration camp survivors (Eitenger, 1973), although these findings are difficult to interpret because of the extent of extreme illness and injury incurred during imprisonment.

Despite the relatively large number of studies that have linked trauma and physical health, not much is known about the longitudinal course of physical reactions to traumatic exposure. Kimerling and Calhoun (1994) found that rape victims reported more physical symptoms than controls 2 weeks, 1

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month, and 4 months post-rape, but not 1 year later; in contrast, the effects of rape on physician visits only emerged at 4 months and continued at 1 year. In Sweden, Bergman and Brismar (1991) also studied utilization in female trauma survivors, comparing medical records for 10 years before and 5 years after a domestic abuse incident. Relative to controls, battered women had increased visits for nontraumatic surgical disorders, gynecological disorders, and medical disorders. Lutgendorf et al. (1995) compared two groups of chronic fatigue syndrome patients, defined by high versus low exposure to the effects of Hurricane Andrew. According to physician diagnosis made within several months after the hurricane, relapse occurred in 67% of the high-exposure group and only 8% of the low-exposure group. Theorell et al. (1992) studied subway drivers who had experienced a "person under train" accident and found that these drivers used more sick days at 3 weeks and 1 year after the accident, relative to control drivers.

In a study with a much longer follow-up interval, Elder et al. (1997) examined physical health trajectories of male participants in the longitudinal Terman study of gifted children, some of whom had served under combat conditions during World War II (WWII). These authors found that combat exposure in WWII predicted a pattern of decline or death from 1945 to 1960, even when physical health in 1945 and other potentially relevant variables were controlled. Combat did not predict a pattern of decline or death across the entire lifespan; its effects were limited to health during the 15 years after the war.

In sum, traumatic exposure is linked to poor physical health outcomes sometimes years after a trauma has occurred. With the exception of the study by Elder and colleagues (1997), the follow-up intervals in longitudinal studies have been quite brief, however. It was in this context that we explored the course of physical symptoms from adulthood to old age in male military veterans of WWII and the Korean conflict.

The Present Study

The Normative Aging Study (NAS; *cf.* Bossé et al., 1984) is a 35-year-old longitudinal study by the Department of Veterans Affairs of 2280 male volunteers who were initially selected for good health. Ninety-five percent of the men are military veterans. Over 40% of these veterans experienced at least some combat (Spiro et al., 1994).

Further analysis of the NAS data can offer a unique longitudinal perspective on the course of physical symptoms as a function of trauma exposure in a cohort of older veterans. A previous study of NAS men found that physical symptoms on the Cornell Medical Index (CMI; Brodman et al., 1956) increased linearly over time (Aldwin et al., 1989). The present study extends this finding by examining the effect of traumatic exposure on the course of symptoms between ages 30 to 75 among veterans of WWII and the Korean conflict. We tested whether type of traumatic exposure (combat only, noncombat only, and both combat and noncombat) was associated with differences in overall amount of symptoms and in rates of change.

Methods

Sample

Participants were drawn from the longitudinal Boston VA Normative Aging Study (Bossé et al., 1984). Enrollment occurred during 1961 to 1970. At study entry, the men ranged in age from 22 to 82 years (M = 42.3, SD = 9.47). As of February 1990, 6% of subjects had been lost to attrition and 16% had died. The sample tends to be of slightly higher socioeconomic status than the national population and consists of an even distribution of white-collar and blue-collar workers. NAS men are comparable to the U.S. population in terms of mental health (Butcher et al., 1991).

The current investigation drew from respondents to a military service questionnaire that was sent to all 1778 study participants who were alive and enrolled in the study as of February 1990. Over 80% responded. The 1210 respondents who had served in either WWII or the Korean conflict were generally comparable to nonrespondents (Spiro et al., 1994). In particular, 43% of respondents and 42% of nonrespondents had combat exposure. From the 1210 men, we selected 1079 who had completed the CMI (Brodman et al., 1956) at least twice during the study and had complete data on the measures described below. Mean age at the time of the military survey was 65.7 years (SD = 6.56; range = 50 to 85). Most men were white (99%; N = 1034), married (89%; N = 798), and had graduated high school (90%; N = 962). At study entry, 56% (N = 651) were employed in blue-collar or service occupations, and the rest held white-collar jobs. At the time of the military survey, 51% were retired (N = 547), 48% were working full- or part-time (N = 515), and 1% (N = 11) were unemployed. (Percentages reflect small amounts of missing data.)

The present sample was comparable to the 131 excluded men except that the present sample was more likely to be: white (99% *vs.* 95%, χ^2 (df = 1, N

= 1162) = 13.3, $p < .001$); WWII era (69% *vs.* 52%, χ^2 (df = 1, N = 1210) = 13.3, $p < .001$); and in slightly better health (on a 5-point scale, M = 4.05 *vs.* 3.82, $t(78.4) = 2.33$, $p < .05$ with correction for unequal variances). Given the small size of differences between groups and their similarity on other measures, we do not believe that omitting the 131 men with incomplete data biased our results.

Measures and Procedure

In the 1990 mail survey described by Spiro et al. (1994), NAS participants completed a military history questionnaire that included questions about combat exposure, posttraumatic stress disorder (PTSD), and depression. In November 1990, the NAS men completed a mail survey that included questions about traumatic life events.

Combat Exposure. We modified Keane et al.'s (1989) 7-item CES by adding two items that pilot-testing had suggested would be useful to capture the experiences of NAS men: "Over how long a period did you serve under combat conditions or subject to enemy action?" and "Even if you were not directly in combat (*e.g.*, a physician), how many times were you exposed to the outcomes of combat (wounded or dead people)?" Individuals who scored > 0 on the modified scale were coded as having combat exposure.

Noncombat Trauma. We assessed lifetime exposure to noncombat trauma with 11 dichotomous items that asked about: personal, familial, and occupational exposure to a serious accident, violent crime, and natural disaster; other personal noncombat trauma; and nonmilitary occupational injury. Specific questions included, "Were you ever in a bad accident, *i.e.*, one in which serious injuries or death occurred?", "Were you ever exposed to a natural disaster, in which you could have been seriously injured or killed?", and "Were any of your loved ones the victims of a bad crime?" We considered a "yes" response to any one of the 11 items as indicating lifetime exposure to noncombat trauma.

Age. Age at the time of each CMI administration was treated as a time-varying variable.

Physical Symptoms. Symptoms were assessed longitudinally with the CMI questionnaire, scored as reported in Aldwin et al. (1989) as the sum of 131 items that assessed different organ systems, *e.g.*, respiratory, cardiovascular. Internal consistency was good: α = .81 for CMI at study entry and .87 for most recent CMI.

Initially, the CMI was administered to participants every 5 years until they reached age 52 and every 3 years thereafter; since 1986, it has been administered every 3 years to all participants. The average length of follow-up was approximately 25 years.

Psychological Symptoms. Psychological correlates of traumatic exposure were examined by using the two scales that were included in the military history survey. PTSD symptoms were measured with the Mississippi Scale for Combat-Related PTSD (Keane et al., 1988; Kulka et al., 1990). Depression symptoms were measured with the Center for Epidemiological Studies-Depression scale (CES-D; Radloff, 1977).

Data Analysis

The analysis of longitudinal data can be challenging, especially if data have varying numbers of measurements per person and varying intervals between measurements both within and between persons. In recent years, several methods have been developed for analyzing such data, including generalized estimating equation (GEE) techniques (Zeger and Liang, 1986) and maximum likelihood methods (Gibbons et al., 1993; Laird and Ware, 1982), both of which account for correlation among repeated measurements on each individual and for heterogeneity in responses among individuals. The GEE method is applicable to almost any type of repeated data—continuous, binary, or counted—and is robust to misspecification of the variance structure. Individuals are not deleted if they have incomplete outcome data, and arbitrary standardization of between-measurement intervals is unnecessary.

We modeled physical symptom change from ages 30 to 75, which meant that each man contributed data to only part of the studied age range. Poisson quasi-likelihood regression was used to predict physical symptoms because the scale was counted data, *i.e.*, 0, 1, 2 symptoms. We exponentiated regression coefficients and standard errors to produce point estimates and confidence intervals for the relative rates in terms of the original scale of measurement. The relative rate for a predictor shows the change associated with a 1-unit change in the predictor, *e.g.*, a rate of 1.09 indicates a 9% increase and a rate of .83 indicates a 17% decrease. Coefficients for age effects are reported per decade.

Group differences in PTSD and depression symptoms were analyzed by ANOVA with Scheffé's procedure to compare group means for significant F-tests.

Results

Sixty-nine percent of the sample (N = 740) had served in WWII. The mean combat exposure score was 6.01 (SD = 8.78, range = 0 to 36). Almost half

TABLE 1
Final Longitudinal Regression Model to Predict Physical Symptoms as a Function of Trauma Exposure

Predictor variable	RR	95% CI
Age (per decade)	1.29***	1.27, 1.31
Noncombat trauma only	1.09	.99, 1.21
Combat exposure only	.96	.85, 1.08
Both combat and noncombat trauma	1.16***	1.05, 1.28

N = 1079; RR = relative rate; 95% CI = 95% confidence interval.
***$p < .001$.

(46%; N = 497) of the men reported at least some combat; the mean among these men was 13.05 (SD = 8.68). Almost two-thirds (63%; N = 678) reported experiencing a noncombat traumatic event during their lifetime. Combining the information about combat and noncombat trauma, we found that 31% of the sample (N = 333) had experienced noncombat trauma only, 14% (N = 152) had experienced combat only, 32% (N = 345) had experienced both types of trauma, and 23% (N = 249) had experienced neither.

Physical symptom scores averaged 9.69 symptoms across both individuals and occasions (SD = 6.80, range = 0 to 61, N = 6968). The number of observations per individual ranged from 2 to 10, but approximately 95% of participants had 5 or more observations. The mean number of observations per individual was 6.45 (SD = 1.65).

In our initial regression model we tested whether: a) physical symptom scores changed linearly with age; b) type of traumatic exposure was associated with differences in symptom levels regardless of age; and c) age-related changes in symptoms varied as a function of type of trauma. The initial model included age as a time-dependent predictor and type of trauma as three fixed predictors; these predictors were coded "0,1" to reflect noncombat only, combat only, and both combat and noncombat trauma, with no trauma serving as the reference category. The initial model also included three time-dependent predictors to test the interaction of age and type of trauma. These terms were not retained in the final model due to a lack of statistical significance.

Table 1 shows the final results of the longitudinal regression analyses. For every decade increment in age, physical symptoms increased 29% ($p < .001$). The experience of either combat or noncombat trauma was not significantly associated with symptom reports. However, the experience of both types of trauma was associated with elevated symptoms across all ages. Men who experienced both combat and noncombat trauma reported 16% more physical symptoms than men who had experienced neither

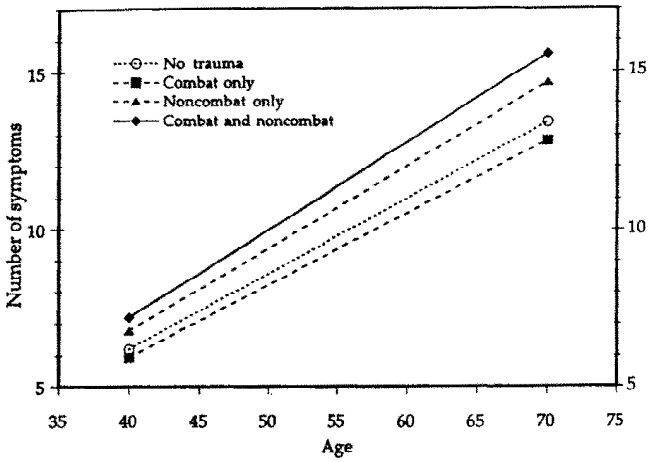

FIG. 1. Predicted physical symptoms as a function of age and type of trauma.

type of trauma ($p < .001$). As noted above, the interactions of age with type of trauma were not statistically significant, and visual inspection of the data suggested no evidence that type of trauma was differentially associated with age-related trajectories.

Figure 1 depicts the effects of age and type of trauma on predicted physical symptoms at ages 40 and 70. The apparent trend for the absolute amount of increase in physical symptoms over time to vary as a function of type of trauma may be confusing given that we did not find an age × type of trauma interaction. This seeming discrepancy is due to the difference between considering the results in relative versus absolute terms. The absence of an interaction means that the rate of age-related change was constant regardless of the type of trauma. But presence of a main effect of type of trauma means that the amount of age-related change will become progressively larger with increasing age because the 29% per decade increase is 29% of a figure that differs as a function of type of trauma, *e.g.*, 16% for men with both combat and noncombat trauma, in contrast with 9% for men with noncombat trauma only.

In an attempt to understand the psychological characteristics of participants in each of the four trauma groups, we examined group differences in PTSD and depression symptoms. Group means for these measures are presented in Table 2. ANOVA showed that the groups differed in PTSD symptoms (F = 12.24, df = 3,1075, $p < .001$) but not in depression symptoms (F = 1.54, df = 3,1068, $p < .20$). Results of a Scheffé test used to compare group means in PTSD symptoms showed that the group that had experienced both combat and noncombat

TABLE 2
PTSD and Depression Symptoms as a Function of Trauma Exposure

	PTSD		Depression	
	Mean	SD	Mean	SD
No trauma	55.83_a	7.97	6.81	6.32
Noncombat only	56.52_a	8.90	6.80	6.23
Combat only	58.22_{ab}	10.31	7.24	5.97
Both combat and noncombat trauma	60.21_b	11.49	7.74	6.88

Column means not sharing a common subscript differ from each other by Scheffé test at $p < .05$; $N = 1079$ for PTSD and 1072 for depression.

trauma differed from the no trauma group and the noncombat only group. Note that average PTSD symptom levels were low and few people had scores ≥ 89, the recommended cutoff score for community-residing veterans (Kulka et al., 1990): 1 in the combat only group and 4 in the combined trauma group.

Discussion

We modeled the course of physical symptoms from ages 30 to 75 among older male veterans as a function of type of trauma exposure. Symptoms increased 29% per decade. These results are comparable to previous longitudinal findings using a larger sample of NAS men and a different analytic technique (Aldwin et al., 1989).

The experience of either combat or noncombat trauma was not associated with increased symptom scores. However, the experience of both types of trauma was associated with 16% higher symptoms levels across all ages studied. In general, our findings are consistent with numerous reports of associations between trauma exposure and negative health outcomes (Friedman and Schnurr, 1995). It is unclear why neither combat or noncombat trauma alone was associated with higher physical symptom scores. One possibility is that we were not able to ascertain whether any given exposure met DSM-IV criteria (American Psychiatric Association, 1994) and, thus, that some of the exposed men did not experience their events as traumatic. The combined category, due to the multiple nature of the traumatic exposure, may have been more likely than either single category to reflect sufficient event severity.

The rate of change in physical symptoms was constant regardless of type of trauma, although the absolute amount of increase was larger in the combined trauma group, as compared with the no trauma group. This finding is not surprising in light of Elder et al.'s (1997) longitudinal study, in which health trajectories varied as a function of combat exposure in WWII veterans only up until 1960. Because our study began in the 1960s, we were unable to study this potentially critical period. Elder et al. also found that combat was unrelated to health outcomes over the entire lifespan, which is similar to our findings with the present data.

Aldwin (1994; Aldwin and Stokols, 1988) has speculated about the conditions under which stress can precipitate long-term negative outcomes. Aldwin and Stokols proposed that events that affect multiple domains or that engender serious sequelae are more likely than events with more constrained effects to generate a negative adaptive spiral. Although we did not test this hypothesis directly, it is consistent with the fact that only combined trauma was related to increased physical symptoms.

Cohen and Williamson (1991) presented a model of stress and infectious disease in which they posit distress as mediating between stressor exposure and disease. In a similar vein, Friedman and Schnurr (1995) have suggested that PTSD is a major mediator of the relationship between trauma and poor physical health. We could not test this hypothesis because we did not have a measure of PTSD status at study entry. However, analyses conducted to examine correlates of traumatic exposure showed that symptoms of PTSD, but not of depression, were elevated in the combined trauma group. These findings lend indirect support to the possibility that PTSD is a mediator of the effects of trauma on health and suggest that it is not merely increased distress, but PTSD specifically, that is important.

Our study has strengths and weaknesses that must be kept in mind when interpreting the results. One is the use of self-report to measure physical health, but the fact that traumatic exposure is linked not just to self-reported health but also to morbidity and mortality (Friedman and Schnurr, 1995) strengthens our interpretation of self-reports as physical health indicators, however. Another limitation is that we cannot be sure that all of the noncombat traumatic events reported occurred before the assessment of corresponding outcomes during the observation period. The most probable consequence of such occurrences would be to add noise to the data, if we assume that symptoms would increase after an event. Thus, our trauma measure may have obscured the true magnitude of the effects of noncombat trauma on symptoms. Yet another limitation of our study is the CMI (Brodman et al., 1956), which may be insensitive to mild or moderate symptoms because it requires "yes/no" answers. Also, the relative healthiness of our sample may limit the generalizability of our findings to more impaired samples. Likewise, the low minority representation in the NAS may limit the generalizability to nonwhite pop-

ulations. We note that the percentage of men in our sample who had experienced some trauma, 77%, is somewhat higher than the 60% to 69% previously reported for community samples (*e.g.*, Kessler et al., 1995; Norris, 1992), but ours is likely higher because of the high proportion of WWII combat veterans.

One strength of our study is our long-term follow-up of approximately 25 years. Only one longitudinal study of physical symptoms of trauma survivors had a follow-up interval longer than 5 years (Elder et al., 1997). Another strength is our large sample size. A further strength of our study is the method of longitudinal data analysis (Zeger and Liang, 1986), which enabled us to take full advantage of the rich data that the NAS has to offer without needing to omit individuals who have missing outcome data or make arbitrary decisions about standardizing measurement intervals. The newer methods that have evolved for the analysis of longitudinal data can eliminate the need for such questionable, and often difficult, practices (see Diggle et al., 1994; Gibbons et al., 1993).

We modeled individual differences in symptom change by testing interactions of age and type of trauma. We used a fixed effect model because we thought it to be an appropriate first step in our attempt to understand long-term reactions to trauma; fixed effect models are more robust than random effect models to misspecification of the random effects structure and deviations from normal errors. We believe that estimating trajectories for (trauma) subgroups in a fixed effect model is a reasonable way to examine individual differences, although we hope in future studies to be able to model individual trajectories as random effects as well.

Conclusions

Our data, along with results of many other investigators (*e.g.*, Golding et al., 1997; Kulka et al., 1990; Leserman et al., 1996; Walker et al., 1992), show that exposure to a traumatic event can be associated with very long-term adverse effects on physical health. It is important for future research to explore the mechanisms through which trauma is related to physical health. We believe that it is not merely exposure to trauma, but one's reaction to it, that is most likely to predict outcome.

Some readers may be surprised that we take the time to make what to them may seem such an obvious point—that an individual's reaction to a traumatic event predicts outcome. We do so, however, to raise what we believe is an issue with important methodological and theoretical implications. PTSD is only one aspect of an individual's response to trauma, which may include other negative outcomes as well as positive outcomes. Significant advances in the understanding of how individuals are affected by trauma may come from multidimensional assessment of its effects. Ideally, such assessment would take place in the context of longitudinal studies that enable us to view how these effects unfold over time.

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